

Set Name Query
side by side

DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; THES=ASSIGNEE;
PLUR=YES; OP=AND

		<u>Hit Count</u>	<u>Set Name</u>
			result set
<u>L9</u>	L8 and (VEGF and (brain adj extract))	3	<u>L9</u>
<u>L8</u>	L6 same (collagen)	25	<u>L8</u>
<u>L7</u>	L6 same (brain adj extract)	3	<u>L7</u>
<u>L6</u>	(endothelial adj cell) same ((peripheral adj blood) or (buffy adj coat))	822	<u>L6</u>
<u>L5</u>	L4 and (collagen)	6	<u>L5</u>
<u>L4</u>	L3 and ((bovine adj brain) adj extract)	7	<u>L4</u>
<u>L3</u>	L2 and (VEGF)	720	<u>L3</u>
<u>L2</u>	(endothelial adj cells) and ((peripheral adj blood) or (buffy adj coat))	3853	<u>L2</u>
<u>L1</u>	Hebbel-Robert-P\$.in.	1	<u>L1</u>

END OF SEARCH HISTORY

```
### Status: Path 1 of [Dialog Information Services via Modem]
### Status: Initializing TCP/IP using {UseTelnetProto 1 ServiceID pto-dialog)
Trying 31060000009999...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
***** HHHHHHHH SSSSSSS?
### Status: Signing onto Dialog
*****  
ENTER PASSWORD:
***** HHHHHHHH SSSSSSS? *****  
Welcome to DIALOG
### Status: Connected

Dialog level 02.12.60D

Last logoff: 01apr03 15:51:41
Logon file001 04apr03 15:36:57
*** ANNOUNCEMENT ***
***  
--File 515 D&B Dun's Electronic Business Directory is now online
completely updated and redesigned. For details, see HELP NEWS 515.
***  
--File 990 - NewsRoom now contains October 2002 to present records.
File 993 - NewsRoom archive contains 2002 records from January 2002-
September 2002. To search all 2002 records, BEGIN 990,993 or B NEWS2002
***  
--Alerts have been enhanced to allow a single Alert profile to be
stored and run against multiple files. Duplicate removal is available
across files and for up to 12 months. The Alert may be run according
to the file's update frequency or according to a custom
calendar-based schedule. There are no additional prices for these
enhanced features. See HELP ALERT for more information.
***  
--U.S. Patents Fulltext (File 654) has been redesigned with
new search and display features. See HELP NEWS 654 for
information.
***  
--Connect Time joins DialUnits as pricing options on Dialog.
See HELP CONNECT for information.
***  
--CLAIMS/US Patents (Files 340,341, 942) have been enhanced
with both application and grant publication level in a
single record. See HELP NEWS 340 for information.
***  
--SourceOne patents are now delivered to your email inbox
as PDF replacing TIFF delivery. See HELP SOURCE1 for more
information.
***  
--Important news for public and academic
libraries. See HELP LIBRARY for more information.
***  
--Important Notice to Freelance Authors--
See HELP FREELANCE for more information
***  
For information about the access to file 43 please see Help News43.
***  
NEW FILES RELEASED
***Dialog NewsRoom - Current 3-4 months (File 990)
***Dialog NewsRoom - 2002 Archive (File 993)
***Dialog NewsRoom - 2001 Archive (File 994)
***Dialog NewsRoom - 2000 Archive (File 995)
***TRADEMARKSCAN-Finland (File 679)
```

***TRADEMARKSCAN-Norway le 678)
***TRADEMARKSCAN-Sweden (File 675)

UPDATING RESUMED
***Delphes European Business (File 481)

RELOADED
***D&B Dun's Electronic Business Directory (File 515)
***U.S. Patents Fulltext 1976-current (File 654)
***Population Demographics (File 581)
***Kompass Western Europe (File 590)
***D&B - Dun's Market Identifiers (File 516)

REMOVED
***Chicago Tribune (File 632)
***Fort Lauderdale Sun Sentinel (File 497)
***The Orlando Sentinel (File 705)
***Newport News Daily Press (File 747)
***U.S. Patents Fulltext 1980-1989 (File 653)
***TOXNET data is added to ToxFile (F156)

New document supplier
IMED has been changed to INFOTRIE (see HELP OINFOTRI)

>>> Enter BEGIN HOMEBASE for Dialog Announcements <<<
>>> of new databases, price changes, etc. <<<

KWIC is set to 50.
HIGHLIGHT set on as '*'
* * * *

File 1:ERIC 1966-2003/Mar 24
(c) format only 2003 The Dialog Corporation

Set	Items	Description
---	---	-----
Cost	is in DialUnits	
?b	155, 159, 5, 73	
04apr03 15:37:27	User259876	Session D485.1
\$0.36	0.104	DialUnits File1
\$0.36	Estimated cost	File1
\$0.11	TELNET	
\$0.47	Estimated cost	this search
\$0.47	Estimated total	session cost 0.104 DialUnits

SYSTEM:OS - DIALOG OneSearch
File 155: MEDLINE(R) 1966-2003/Mar W5
(c) format only 2003 The Dialog Corp.
***File 155: Medline has been reloaded and accession numbers have changed. Please see HELP NEWS 155.**
File 159: Cancerlit 1975-2002/Oct
(c) format only 2002 Dialog Corporation
***File 159: Cancerlit ceases updating with immediate effect.**
Please see HELP NEWS.
File 5: Biosis Previews(R) 1969-2003/Mar W5
(c) 2003 BIOSIS
***File 5: Alert feature enhanced for multiple files, duplicates removal, customized scheduling. See HELP ALERT.**
File 73: EMBASE 1974-2003/Mar W5
(c) 2003 Elsevier Science B.V.
***File 73: Alert feature enhanced for multiple files, duplicates removal, customized scheduling. See HELP ALERT.**

Set Items Description

?s (endothelial (w) cells) and ((peripheral (w) blood) or (buffy (w) coat))

Processing

Processing

311818 ENDOTHELIAL
4929173 CELLS
169176 ENDOTHELIAL(W) CELLS
951423 PERIPHERAL
4805068 BLOOD
295772 PERIPHERAL(W) BLOOD
5350 BUFFY
44440 COAT
4635 BUFFY(W) COAT
S1 3580 (ENDOTHELIAL (W) CELLS) AND ((PERIPHERAL (W) BLOOD) OR
(BUFFY (W) COAT))

?s s1 and (VEGF)

3580 S1
26385 VEGF

S2 96 S1 AND (VEGF)

?s s2 and (collagen)

96 S2
263221 COLLAGEN

S3 3 S2 AND (COLLAGEN)

?rd

...completed examining records

S4 2 RD (unique items)

?t s4/3,k/all

4/3,K/1 (Item 1 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

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09338938 21097696 PMID: 11166280

Monocytes coexpress endothelial and macrophagocytic lineage markers and form cord-like structures in Matrigel under angiogenic conditions.

Schmeisser A; Garlich C D; Zhang H; Eskafi S; Graffy C; Ludwig J; Strasser R H; Daniel W G

Department of Cardiology, Technical University of Dresden, Heart Center Dresden, Fetscherstr. 76, D-01307, Dresden, Germany.
alexanderschmeis@t-online.de

Cardiovascular research (Netherlands) Feb 16 2001, 49 (3) p671-80,
ISSN 0008-6363 Journal Code: 0077427

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

OBJECTIVES: It has been shown that circulating human non-adherent CD34+ cells coexpressing vascular endothelial growth factor (*VEGF*)-R2 and AC133 have the capacity to differentiate into adherent mature *endothelial* *cells*. However, prior studies have demonstrated that a much bigger subset of primary adherent mononuclear cells can also form endothelial progenitor cells (EPC). To determine the...

... as a firmly adherent and plastic cell type have the potential to differentiate into an endothelial phenotype. **METHODS:** CD34-/CD14+ monocytes were isolated from human *peripheral* *blood* by adherence separation and magnetic bead selection (purity >90%) and cultured on fibronectin-coated plastic dishes (medium containing *VEGF* 10 ng/ml, basic fibroblast growth factor (bFGF) 2 ng/ml, insulin like growth factor (IGF-1) 1 ng/ml, 20% fetal calf serum). **RESULTS...**

...; AN; Biological Markers--analysis--AN; Cadherins--analysis--AN; Cell Adhesion--drug effects--DE; Cell Differentiation--drug effects--DE; Cell Division--drug effects--DE; Cells, Cultured; *Collagen*; Drug Combinations; Endothelial Growth Factors--pharmacology--PD; Fibroblast Growth Factor 2 --pharmacology--PD; Flow Cytometry; Laminin; Lymphokines--pharmacology--PD; Monocytes--drug effects--DE; Nitric-Oxide...

...Chemical Name: Endothelial Growth Factors; Growth Substances; Laminin; Lymphokines; Proteoglycans; Receptors, Growth Factor; cadherin 5; vascular endothelial growth factor; von Willebrand Factor; Fibroblast Growth Factor 2; matrigel; *Collagen*; endothelial constitutive nitric oxide synthase; Nitric-Oxide Synthase; Receptor Protein-Tyrosine Kinases; Receptors, Vascular Endothelial Growth Factor

4/3, K/2 (Item 1 from file: 5)
DIALOG(R) File 5:Biosis Previews(R)
(c) 2003 BIOSIS. All rts. reserv.

13557839 BIOSIS NO.: 200200186660

Conditioned media from CLL and SLL stimulates in vitro endothelial cell proliferation and decreases endostatin generation by *endothelial* *cells*, both likely mediated by bFGF.

AUTHOR: Rimsza Lisa M(a); Pastos Karen M(a); Lynch James W; Braylan Raul C (a)

AUTHOR ADDRESS: (a)Pathology, Immunology and Laboratory Medicine, University of Florida, Gainesville, FL**USA

JOURNAL: Blood 98 (11 Part 1):p361a November 16, 2001

MEDIUM: print

CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001

ISSN: 0006-4971

RECORD TYPE: Abstract

LANGUAGE: English

Conditioned media from CLL and SLL stimulates in vitro endothelial cell proliferation and decreases endostatin generation by *endothelial* *cells*, both likely mediated by bFGF.

ABSTRACT: We investigated the cellular source of increased serum basic fibroblastic growth factor (bFGF) and vascular endothelial growth factor (*VEGF*) reported in patients with chronic lymphocytic leukemia (CLL). We also explored the effects of tumor-secreted bFGF on endothelial cell proliferation and production of endostatin (a proteolytic fragment of *collagen* XVIII generated by *endothelial* *cells* from basement membrane material). We first cultured unstimulated mononuclear cells from 4 *peripheral* *blood* (PB) CLL and 5 lymph node (LN) samples of small lymphocytic lymphoma (SLL), 2 normal PB mononuclear cells, and 2 reactive LN samples. The conditioned media (CM) was assayed for secreted bFGF and *VEGF*, then used in a 72 hour in vitro HUVEC proliferation assay. bFGF was detected in CM from 2 of 4 PB CLL samples and 5...

...samples. HUVEC proliferation in the presence of CM was variable but proportional to secreted levels of bFGF, a correlation most pronounced in the LN samples. *VEGF* was not detected in CM from any of the mononuclear cell populations, although it was secreted by adherent cell layers isolated from 3 LN samples. HUVEC secreted neither *VEGF* nor bFGF. We next cultured HUVEC in CM from 3 CLL (1 PB, 1 pleural fluid, 1 bone marrow), 2 LN with SLL, and 1...

...may originate from the tumor cells themselves and may be responsible for increased angiogenesis in the BM and LN of these patients, while increased serum *VEGF* may be secreted by another cellular source such as lymph node adherent cells. Our findings also indicate that CM from human CLL and SLL samples influence endostatin generation by *endothelial* *cells*, an effect that may be mediated by bFGF. The mechanism of interaction between bFGF and endostatin might be an important direction in anti-angiogenic investigation.

DESCRIPTORS:

...ORGANISMS: PARTS ETC: *peripheral* *blood* mononuclear cell

CHEMICALS & BIOCHEMICALS: ...*collagen* XVIII...

...vascular endothelial growth factor (*VEGF*)--

?ds

Set	Items	Description
S1	3580	(ENDOTHELIAL (W) CELLS) AND ((PERIPHERAL (W) BLOOD) OR (BU- FFY (W) COAT))
S2	96	S1 AND (VEGF)
S3	3	S2 AND (COLLAGEN)
S4	2	RD (unique items)
?s s2 and (brain (w) extract)	96	S2
	1557817	BRAIN
	226107	EXTRACT
	1957	BRAIN(W) EXTRACT
S5	0	S2 AND (BRAIN (W) EXTRACT)
?s s2 and (cobblestone)	96	S2
	2291	COBBLESTONE
S6	0	S2 AND (COBBLESTONE)
?s s2 and (proliferation or expansion)	96	S2
	537141	PROLIFERATION
	124103	EXPANSION
S7	36	S2 AND (PROLIFERATION OR EXPANSION)
?rd		...completed examining records
	S8	16 RD (unique items)

?t s8/3,k/all

8/3,K/1 (Item 1 from file: 155)

DIALOG(R)File 155: MEDLINE(R)

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09853054 21665274 PMID: 11806246

Soluble VEGFR-1 secreted by *endothelial* *cells* and monocytes is present in human serum and plasma from healthy donors.

Barleon B; Reusch P; Totzke F; Herzog C; Keck C; Martiny-Baron G; Marme D
RELIATEch GmbH, Mascheroderweg 1b, D-38124 Braunschweig, Germany.
Bernhard.Barleon@reliatech.de

Angiogenesis (Netherlands) 2001, 4 (2) p143-54, ISSN 0969-6970

Journal Code: 9814575

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Soluble VEGFR-1 secreted by *endothelial* *cells* and monocytes is present in human serum and plasma from healthy donors.

... present in serum of pregnant women. The aim of the present study was to investigate the presence of this endogenous vascular endothelial growth factor-A (*VEGF*-A) antagonist in human serum in more detail. sVEGFR-1 was detected in human serum and plasma from normal healthy male and female donors by ELISA. sVEGFR-1 levels ranged from non-detectable up to 440 pg/ml, with no significant difference between male and female donors. In addition, vein *endothelial* *cells* (ECs) from an intact vascular bed, the umbilical cord, were shown to secrete sVEGFR-1. Furthermore, human *peripheral* *blood* monocytes, a non-EC type expressing VEGFR-1, were shown to contribute to the sVEGFR-1 detectable in human serum and plasma for the first time. EC- and monocyte-derived sVEGFR-1 proved capable of inhibiting the *VEGF*-induced *proliferation* and migration of ECs in vitro. Finally, secretion of sVEGFR-1 was increased by the angiogenic factor basic fibroblast growth factor (bFGF) in human ECs and was also enhanced in lipopolysaccharide-activated human monocytes. In human umbilical vein *endothelial* *cells*, both the membrane-bound and the sVEGFR-1 seem to be equally regulated on the mRNA as well as the protein level. The presence of...

... sVEGFR-1 in human serum and plasma of normal male and female donors strongly suggests that it plays an important role as a naturally occurring *VEGF* antagonist in the regulation and availability of *VEGF*-mediated biological activities in vivo.

8/3,K/2 (Item 2 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

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09739563 21541282 PMID: 11684862

Bioactivity of the vascular endothelial growth factor trapped in fibrin clots: production of IL-6 and IL-8 in monocytes by fibrin clots.

Tezono K; Sarker K P; Kikuchi H; Nasu M; Kitajima I; Maruyama I
Second Department of Internal Medicine, Oita Medical University, Oita, Japan.

Haemostasis (Switzerland) Mar-Apr 2001, 31 (2) p71-9, ISSN 0301-0147 Journal Code: 0371574

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

...growth factors such as platelet-derived growth factor and transforming growth factor beta. In the present study, we demonstrated that the vascular endothelial growth factor (*VEGF*) could be bound to fibrin clots in the plasma, and that incubation of the *endothelial* *cells* with these *VEGF*-bound fibrin clots induced *proliferation* of *endothelial* *cells*. Thus, it suggests that clot-bound *VEGF* may play a role in wound healing through the *proliferation* of *endothelial* *cells* and vascular smooth-muscle cells. On the other hand, a noticeable migration of monocytes was observed when they were cultured on dishes in the presence of *VEGF*-bound fibrin clots. Moreover, *peripheral* *blood* monocytes incubated in the presence of *VEGF* -bound fibrin clots strikingly increased the production of IL-6 and IL-8, demonstrating that *VEGF* trapped in fibrin clots not only induces *proliferation* of human umbilical vein *endothelial* *cells* and migration of monocytes but also enhances secretion of IL-6 and IL-8. Thus, our data suggest that fibrin clots that contain several growth...

8/3,K/3 (Item 3 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

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09725852 21521211 PMID: 11641248

Fibrocytes induce an angiogenic phenotype in cultured *endothelial* *cells* and promote angiogenesis in vivo.

Hartlapp I; Abe R; Saeed R W; Peng T; Voelter W; Bucala R; Metz C N
Laboratory of Vascular Biology, The Picower Institute for Medical Research, Manhasset, New York 11030, USA.

FASEB journal - official publication of the Federation of American Societies for Experimental Biology (United States) Oct 2001, 15 (12) p2215-24, ISSN 1530-6860 Journal Code: 8804484

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Fibrocytes induce an angiogenic phenotype in cultured *endothelial* *cells* and promote angiogenesis in vivo.

... years have identified several growth factors, cytokines, and enzymes that promote blood vessel formation. Most have revealed how individual factors promote an angiogenic phenotype in *endothelial* *cells* in vitro or contribute to blood vessel formation in vivo. However, the fundamental question that remains unanswered is how the cellular microenvironment contributes to angiogenesis. Fibrocytes are a recently characterized

mesenchymal cell type isolated from *peripheral* *blood* that rapidly enter subcutaneously implanted wound chambers and sites of tissue injury. Here we describe the induction of an angiogenic phenotype in microvascular *endothelial* *cells* in vitro and promotion of angiogenesis in vivo by cultured fibrocytes. Fibrocytes constitutively secrete extracellular matrix-degrading enzymes, primarily matrix metalloproteinase 9, which promotes endothelial cell invasion. In addition, fibrocytes secrete several proangiogenic factors including *VEGF*, bFGF, IL-8, PDGF, and hematopoietic growth factors that promote endothelial cell migration, *proliferation*, and/or tube formation. By contrast, they do not produce representative antiangiogenic factors. Finally, both autologous fibrocytes and fibrocyte-conditioned media were found to induce...

8/3,K/4 (Item 4 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

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09694424 21486847 PMID: 11600818

[Angiogenesis in patients with hematologic malignancies]

Angiogenese bei hamatologischen Neoplasien.

Mesters R M; Padro T; Steins M; Bieker R; Retzlaff S; Kessler T; Kienast J; Berdel W E

Medizinische Klinik und Poliklinik A, Universitätsklinikum Münster, Germany. meesters@uni-muenster.de

Onkologie (Switzerland) Sep 2001, 24 Suppl 5 p75-80, ISSN 0378-584X
Journal Code: 7808556

Document type: Journal Article; Review; Review, Tutorial ; English
Abstract

Languages: GERMAN

Main Citation Owner: NLM

Record type: Completed

...chemotherapy. Tumor angiogenesis depends on the expression of specific mediators that initiate a cascade of events leading to the formation of new microvessels. Among these, *VEGF* (vascular endothelial growth factor), FGF (fibroblast growth factor) and angiopoietins play a pivotal role in the induction of neovascularization in solid tumors. These cytokines stimulate migration and *proliferation* of *endothelial* *cells* and induce angiogenesis in vivo. Recent data suggest an important role for these mediators in hematologic malignancies as well. Isolated AML blasts overexpress *VEGF* and *VEGF* receptor 2. Thus, the *VEGF*/VEGFR-2 pathway can promote the growth of leukemic blasts in an autocrine and paracrine manner. Therefore, neovascularization and angiogenic mediators/receptors may be promising...

...infiltration in the bone marrow). This was accompanied by an increase in platelet counts and hemoglobin values. One additional patient showed a significant improvement of *peripheral* *blood* counts without fulfilling the criteria of a PR. In parallel, we observed a significant decrease in microvessel density in these 5 patients during treatment with thalidomide. In conclusion, thalidomide seems to have anti-angiogenic as well as anti-leukemic activity in AML. The *VEGF*/VEGFR-2 pathway seems to play an important role in AML. Therefore, receptor tyrosine kinase inhibitors like SU5416 or SU6668 are currently evaluated in the...

8/3,K/5 (Item 5 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

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09612594 21397897 PMID: 11506504

Enhanced *VEGF* production and decreased immunogenicity induced by TGF-beta 1 promote liver metastasis of pancreatic cancer.

Teraoka H; Sawada T; Nishihara T; Yashiro M; Ohira M; Ishikawa T; Nishino H; Hirakawa K

First Department of Surgery, Osaka City University Medical School, 1-4-3 Asahi-machi, Abeno-ku, Osaka 545-8585, Japan.

British journal of cancer (Scotland) Aug 17 2001, 85 (4) p612-7,
ISSN 0007-0920 Journal Code: 0370635

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Enhanced *VEGF* production and decreased immunogenicity induced by TGF-beta 1 promote liver metastasis of pancreatic cancer.

... potential by in vivo splenic injection with TGF-beta1. Consequently, we examined the role of TGF-beta1 on in vitro angiogenesis and received cytotoxicity by *peripheral* *blood* mononuclear leukocytes (PBMLs). While TGF-beta1 slightly decreased cell *proliferation*, it also upregulated *VEGF* production in all cancer cells examined. The binding of PBMLs to cancer cells and cancer cell cytotoxicity during co-culture with PBMLs were remarkably decreased...

... metastasis despite their high immunogenetic and angiogenetic abilities, which was attributed to a lack of expression of the cell surface carbohydrates that induce attachment to *endothelial* *cells*. We concluded that the presence of TGF-beta1 in the microenvironment of tumour site might play an important role in enhancing liver metastasis of pancreatic...

8/3,K/6 (Item 6 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

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09217028 20526948 PMID: 11076319

[Angiogenesis and vasculogenesis. Therapeutic strategies for stimulation of postnatal neovascularization]

Angiogenese und Vaskulogenese. Therapeutische Strategien zur Stimulation der postnatalen Neovaskularisation.

Kalka C; Asahara T; Krone W; Isner J M

Department of Medicine (Cardiovascular Research), Tufts University School of Medicine, St. Elizabeth's Medical Center, Boston, Massachusetts, USA. Ckalka@juno.com

Herz (GERMANY) Sep 2000, 25 (6) p611-22, ISSN 0340-9937
Journal Code: 7801231

Document type: Journal Article; Review; Review, Tutorial ; English
Abstract

Languages: GERMAN

Main Citation Owner: NLM

Record type: Completed

... factors could promote collateral artery development in animal models of peripheral and myocardial ischemia. Subsequent clinical trials using gene transfer of naked DNA encoding for *VEGF* for the treatment of critical limb and myocardial ischemia documented the safety and clinical benefit of this novel therapeutic approach. Several objective methods indicated marked improvement in collateral vessel development. Vasculogenesis describes the development of new blood vessels from in situ differentiating *endothelial* *cells*. Recently considered to be restricted to embryogenesis, there exists now striking evidence that endothelial progenitor cells (EPC) circulate also in adult *peripheral* *blood* able to participate in ongoing neovascularization. Different cytokines and growth factors have a stimulatory effect on these bone-marrow derived EPC. Granulocyte macrophage colony stimulating factor (GM-CSF) and vascular endothelial growth factor (*VEGF*) mobilize EPC from the bone marrow into the peripheral circulation. While their endogenous contribution to postnatal neovascularization needs to be documented, the iatrogenic *expansion* and mobilization of EPC might represent an effective means to augment the resident population of *endothelial* *cells* (ECs). This kind of cell therapy for tissue regeneration in ischemic cardiovascular diseases

opens a novel and challenging clinical option besides or in addition to...

8/3,K/7 (Item 7 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

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09046073 20341172 PMID: 10880756

Expression and secretion of vascular endothelial growth factor-A by cytokine-stimulated hematopoietic progenitor cells. Possible role in the hematopoietic microenvironment.

Bautz F; Rafii S; Kanz L; Mohle R

Department of Medicine II, University of Tubingen, Tubingen, Germany.

Experimental hematology (NETHERLANDS) Jun 2000, 28 (6) p700-6,

ISSN 0301-472X Journal Code: 0402313

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

In the hematopoietic microenvironment, bone marrow *endothelial* *cells* may play an important role in trafficking and maintenance of progenitor and stem cells due to adhesive interactions and paracrine secretion of hematopoietic growth factors. However, it is unknown whether progenitors in turn modulate endothelial *proliferation* and function. We analyzed mRNA expression (Northern blot) and release of vascular endothelial growth factor-A (*VEGF*-A), which specifically acts on *endothelial* *cells*, by cytokine-stimulated *peripheral* *blood* -derived CD34+ hematopoietic progenitor cells. While unstimulated CD34+ cells expressed *VEGF*-A mRNA weakly without cytokine release in vitro, incubation for 24 hours with a single cytokine (e.g., kit ligand [KL]) resulted in increased *VEGF*-A mRNA expression and significant secretion of *VEGF*-A into the supernatant. The amount of *VEGF* released was substantially augmented by incubation with a combination of cytokines (e.g., KL, IL-3, GM-CSF, G-CSF), or by exposure to hematopoietic cytokines for a longer time period. In addition, we show that *VEGF* induced the release of hematopoietic growth factors (GM-CSF) by bone marrow *endothelial* *cells* and that in vitro stromal cell-derived factor-1 (SDF-1) driven transendothelial progenitor cell migration was increased by the presence of *VEGF*, which might be due to pore formation (increased endothelial fenestration). In vivo, release of *VEGF* by progenitor cells may result in a paracrine loop supporting *proliferation* of both endothelium and progenitors and may facilitate transendothelial migration during cytokine-induced progenitor cell mobilization.

8/3,K/8 (Item 1 from file: 159)

DIALOG(R) File 159: Cancerlit

(c) format only 2002 Dialog Corporation. All rts. reserv.

02414367 PMID: 98639763

Production of angiogenic factors and plasminogen degrading enzymatic activity by human ovarian cancer cells (Meeting abstract).

Department of Obstetrics and Gynecology, The University of Texas Medical Branch, Galveston, TX 77555-0587, USA

Proc Annu Meet Am Assoc Cancer Res 1997, 38, ISSN 0197-016X

Document Type: MEETING ABSTRACTS

Languages: ENGLISH

Main Citation Owner: NOTNLM

Record type: Completed

Tumor angiogenesis represents a cascade of cellular processes involving *endothelial* *cells*. We studied the production of tumor derived angiogenic factors (TAF) and the plasminogen degrading enzyme activity (PDEA) by epithelial cells derived from ovarian tumors (FIGO, stages I-IV, n=15) and ascitic fluid (n=8). The serum free tumor conditioned medium (TCM) collected from mononuclear cells derived from *peripheral* *blood*

(n=15) and ascitic fluid (n=8) of ovarian cancer patients produced TAF and PDEA. The presence TAF and PDEA was also studied from concentrated peritoneal ascitic fluid (n=8) collected from ovarian cancer patients. Multiple in vitro (ELISA, MTT-*proliferation* and Boyden chamber and wound healing migration assays) and in vivo (corneal implant assay) angiogenesis related assays were used to study the presence of angiogenic...

... close to the molecular weight of angiostatin. The angiogenic factors identified by ELISA in the TCM were IL-2, IL-6, IL-8, beta-FGF, *VEGF*, angiogenin, GM-CSF and TNF-alpha. Thus it can be concluded that the epithelial cells derived from tumors and ascitic fluid and mononuclear cells from *peripheral* *blood* and ascitic fluid from ovarian cancer patients produce excessive amounts of a variety of angiogenic factors besides plasminogen degrading enzymes.

8/3,K/9 (Item 1 from file: 5)
DIALOG(R) File 5:Biosis Previews(R)
(c) 2003 BIOSIS. All rts. reserv.

13557839 BIOSIS NO.: 200200186660

Conditioned media from CLL and SLL stimulates in vitro endothelial cell *proliferation* and decreases endostatin generation by *endothelial* *cells*, both likely mediated by bFGF.

AUTHOR: Rimsza Lisa M(a); Pastos Karen M(a); Lynch James W; Braylan Raul C (a)

AUTHOR ADDRESS: (a)Pathology, Immunology and Laboratory Medicine, University of Florida, Gainesville, FL**USA

JOURNAL: Blood 98 (11 Part 1):p361a November 16, 2001

MEDIUM: print

CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001

ISSN: 0006-4971

RECORD TYPE: Abstract

LANGUAGE: English

Conditioned media from CLL and SLL stimulates in vitro endothelial cell *proliferation* and decreases endostatin generation by *endothelial* *cells*, both likely mediated by bFGF.

ABSTRACT: We investigated the cellular source of increased serum basic fibroblastic growth factor (bFGF) and vascular endothelial growth factor (*VEGF*) reported in patients with chronic lymphocytic leukemia (CLL). We also explored the effects of tumor-secreted bFGF on endothelial cell *proliferation* and production of endostatin (a proteolytic fragment of collagen XVIII generated by *endothelial* *cells* from basement membrane material). We first cultured unstimulated mononuclear cells from 4 *peripheral* *blood* (PB) CLL and 5 lymph node (LN) samples of small lymphocytic lymphoma (SLL), 2 normal PB mononuclear cells, and 2 reactive LN samples. The conditioned media (CM) was assayed for secreted bFGF and *VEGF*, then used in a 72 hour in vitro HUVEC *proliferation* assay. bFGF was detected in CM from 2 of 4 PB CLL samples and 5 of 5 LN with SLL samples but none of the normal PB or reactive LN samples. HUVEC *proliferation* in the presence of CM was variable but proportional to secreted levels of bFGF, a correlation most pronounced in the LN samples. *VEGF* was not detected in CM from any of the mononuclear cell populations, although it was secreted by adherent cell layers isolated from 3 LN samples. HUVEC secreted neither *VEGF* nor bFGF. We next cultured HUVEC in CM from 3 CLL (1 PB, 1 pleural fluid, 1 bone marrow), 2 LN with SLL, and 1 normal PB mononuclear cell suspension and measured HUVEC *proliferation* and endostatin generation by ELISA assay at the end of 72 hours. HUVEC generation of endostatin in response to CM was inversely proportional to the effect on endothelial *proliferation*. In addition, the average endostatin concentration generated by HUVEC was higher (120+/-9 pg/ml) in the 3 cases (including normal PB) when the CM ...

...3 CM containing bFGF ($p=0.01$), indicating an inverse relationship between bFGF in CM and endostatin generation by HUVEC. Added recombinant bFGF increased HUVEC *proliferation* while decreasing endostatin generation confirming the above. These results suggest that increased serum bFGF reported in patients with CLL may originate from the tumor cells themselves and may be responsible for increased angiogenesis in the BM and LN of these patients, while increased serum *VEGF* may be secreted by another cellular source such as lymph node adherent cells. Our findings also indicate that CM from human CLL and SLL samples influence endostatin generation by *endothelial* *cells*, an effect that may be mediated by bFGF. The mechanism of interaction between bFGF and endostatin might be an important direction in anti-angiogenic investigation.

DESCRIPTORS:

...ORGANISMS: human umbilical vein endothelial cell, *proliferation*;
...ORGANISMS: PARTS ETC: circulatory system, *proliferation*; ...

...*peripheral* *blood* mononuclear cell

CHEMICALS & BIOCHEMICALS: ...vascular endothelial growth factor {*VEGF*}
}--

8/3, K/10 (Item 2 from file: 5)

DIALOG(R) File 5:Biosis Previews(R)

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13522722 BIOSIS NO.: 200200151543

New-found regulators of angiogenesis: Platelet- and tumor-cell-derived microparticles.

AUTHOR: Janowska-Wieczorek Anna; Majka Marcin(a); Kijowski Jacek; Libura Jola; Marquez Leah; Zhao Dongling; Ross Lisa; Kawa Milosz(a); Ratajczak Mariusz Z(a)

AUTHOR ADDRESS: (a)James Graham Brown Cancer Center, Univ. of Louisville, Louisville, KY**USA

JOURNAL: Blood 98 (11 Part 2):p54b November 16, 2001

MEDIUM: print

CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 2 Orlando, Florida, USA December 07-11, 2001

ISSN: 0006-4971

RECORD TYPE: Abstract

LANGUAGE: English

...ABSTRACT: eukaryocytic cells or secreted as exosomes. MP in the environment of the growing tumor may be derived from i) the tumor cells by shedding, ii) *peripheral* *blood* (PB) platelets activated by tumor cells and iii) tumor-infiltrating lymphocytes, monocytes and basophils. We hypothesize that MP are important but under-appreciated components of ...

...biological effects of MP in tumorigenesis and angiogenesis we isolated and purified MP from tumor cells and activated PB platelets and subsequently exposed tumor and *endothelial* *cells* (HUVEC) to them. We observed that MP stimulated phosphorylation of MAPK p42/44, activated the PI-3K-AKT axis in several tumor cell lines and significantly increased the secretion of the angiogenic factors *VEGF* and FGF-2. Moreover, incubation of these cell lines with platelet-derived MP modified the activities of the matrix metalloproteinases (MMPs), necessary for endothelial cell migration and *proliferation*, that they secreted. We found after exposure to MP the active form of MMP-2 (which was inhibited by o-phenanthroline) in several neuroblastoma (6 out of 6 cell lines) and rhabdomyosarcoma (5/6) cell lines. Furthermore, MP derived from tumor cell lines chemo-attracted HUVEC directly and stimulated their *proliferation* in vitro. Generally, the biological effects of PMPs were only partly reduced by heat inactivation or trypsin digest, indicating that, in addition to the protein...

...that MP play an important role in angiogenesis by i) stimulating secretion of angiogenic factors by tumor cells, ii) activating MMP-2, and iii) stimulating *endothelial* *cells* directly. A better understanding of the role MP play in angiogenesis could help us to develop novel therapeutic anti-angiogenic approaches for treatment of various...

DESCRIPTORS:

...ORGANISMS: human umbilical vein *endothelial* *cells*

...ORGANISMS: PARTS ETC: *peripheral* *blood* {PB}

CHEMICALS & BIOCHEMICALS: ...*VEGF* {vascular endothelial growth factor}

8/3,K/11 (Item 3 from file: 5)
DIALOG(R) File 5:Biosis Previews(R)
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13516111 BIOSIS NO.: 200200144932

The angiogenic response to skeletal injury is preserved in the elderly.

AUTHOR: Street J T(a); Wang J H; Wu Q D; Wakai A; McGuinness A; Redmond H P

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JOURNAL: Journal of Orthopaedic Research 19 (6):p1057-1066 November, 2001

MEDIUM: print

ISSN: 0736-0266

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

...ABSTRACT: results in local (circulating) and systemic (fracture site) 'angiogenic' responses and that these reparative mechanisms are attenuated with advanced patient age. This prospective study examined *peripheral* *blood* and fracture hematoma from 32 patients, 16 under 40 years and 16 over the age of 75, undergoing emergent surgery for isolated fracture. The angiogenic cytokines vascular endothelial growth factor (*VEGF*) and platelet-derived growth factor (PDGF) were assayed. Endothelial cell cultures were supplemented with patient plasma and fracture hematoma and angiogenesis determined in vitro by measuring cell *proliferation* and blood vessel tube formation. Angiogenesis was determined in vivo using a murine dorsal wound pocket model and quantification of new blood vessel formation after 7 days. We found that all injured patients, irrespective of age, have elevated plasma and fracture hematoma levels of *VEGF* and PDGF. These elevated cytokine concentrations translate into biologically significant angiogenic effects, in vitro and in vivo. These effects are primarily *VEGF* mediated and are not dependent on patient age. The biological activity of these growth factors does not diminish with advanced age. Thus skeletal injury does...

DESCRIPTORS:

...ORGANISMS: PARTS ETC: *endothelial* *cells*--...

...*peripheral* *blood*--

CHEMICALS & BIOCHEMICALS: ...vascular endothelial growth factor {*VEGF* }

MISCELLANEOUS TERMS: ...cell *proliferation*;

8/3,K/12 (Item 4 from file: 5)
DIALOG(R) File 5:Biosis Previews(R)
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13104761 BIOSIS NO.: 200100311910

In vivo inhibition of matrix metalloproteinases blocks chemo-cytokine induced endothelial and hematopoietic stem cell mobilization.

AUTHOR: Heissig B(a); Hattori K(a); Dias S(a); Crystal R G(a); Wood J; Moore M A S(a); Rafii S(a)

AUTHOR ADDRESS: (a)Div. of Hematol-Oncol., Cornell U. Med. College, New

York, NY**USA

JOURNAL: Blood 96 (11 Part 1):p540a November 16, 2000

MEDIUM: print

CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of Hematology San Francisco, California, USA December 01-05, 2000

SPONSOR: American Society of Hematology

ISSN: 0006-4971

RECORD TYPE: Abstract

LANGUAGE: English

SUMMARY LANGUAGE: English

...ABSTRACT: mobilization of hematopoietic stem cells (HSC) and circulating endothelial precursor cells (CEPs) involves interactions with adhesion molecules and chemokines. The chemocytokines, GCSF, SDF-1 and *VEGF* have been shown to play a key role in the trafficking of HSCs and CEPs. These chemocytokines induce expression and activation of metalloproteinases (MMPs) in...

...hypothesized that in vivo inhibition of MMPs may abrogate chemocytokine-induced mobilization of HSCs. We have shown that adenoviral vectors expressing SDF-1 (AdSDF1) and *VEGF* (AdVEGF) induced mobilization of HSCs and CEPs to the *peripheral* *blood* in mice. Intravenous injection of AdVEGF, AdSDF1 or subcutaneous (SC) injection of GCSF into mice results in a 3 fold increase in white blood cell...

...presence of MPI. No effect on WBC and CFUs was found when MPI or AdNull was given alone. To test whether MPI inhibits stem cell *proliferation* induced by SDF-1 and *VEGF*, BM cellularity and BM CFC were assessed. In mice injected with MPI, the number of BM cells increased at least 2 fold, compared to those...

...in AdVEGF treated as compared to AdNull treated mice, and this could be further augmented by additional treatment with MPI. Stem cell mobilization rather than *proliferation* seems to depend on MMP activation. CEPs with late outgrowth potential, were significantly increased in AdSDF1, AdVEGF and GCSF treated animals. Simultaneous injection of MPI reduced the number of CEPs by 4.5 fold in AdSDF1 and GCSF treated mice. These data indicate that MMP activation by SDF-1, *VEGF* and GCSF, is essential for in vivo mobilization of HSCs and CEPs.

DESCRIPTORS:

...ORGANISMS: PARTS ETC: *endothelial* *cells*--

CHEMICALS & BIOCHEMICALS: ...*VEGF* {vascular endothelial growth factor}

8/3,K/13 (Item 5 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

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12763781 BIOSIS NO.: 200000517404

Angiogenesis and vasculogenesis. Therapeutic approaches for stimulation of post-natal neovascularization.

AUTHOR: Kalka Christoph(a); Asahara Takayuki; Krone Wilhelm; Isner Jeffrey M

AUTHOR ADDRESS: (a)Cardiovascular Research, St. Elizabeth's Medical Center, 736 Cambridge Street, Boston, MA, 02135**USA

JOURNAL: Herz 25 (6):p611-622 September, 2000

MEDIUM: print

ISSN: 0340-9937

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: German; Non-English

SUMMARY LANGUAGE: English; German

...ABSTRACT: factors could promote collateral artery development in animal models of peripheral and myocardial ischemia. Subsequent clinical trials using gene transfer of naked DNA encoding for *VEGF* for the treatment of

critical limb and myocardial ischemia documented the safety and clinical benefit of this novel therapeutic approach. Several objective methods indicated marked improvement in collateral vessel development. Vasculogenesis describes the development of new blood vessels from in situ differentiating *endothelial* *cells*. Recently considered to be restricted to embryogenesis, there exists now striking evidence that endothelial progenitor cells (EPC) circulate also in adult *peripheral* *blood* able to participate in ongoing neovascularization. Different cytokines and growth factors have a stimulatory effect on these bone-marrow derived EPC. Granulocyte macrophage colony stimulating factor (GM-CSF) and vascular endothelial growth factor (*VEGF*) mobilize EPC from the bone marrow into the peripheral circulation. While their endogenous contribution to postnatal neovascularization needs to be documented, the iatrogenic *expansion* and mobilization of EPC might represent an effective means to augment the resident population of *endothelial* *cells* (ECs). This kind of cell therapy for tissue regeneration in ischemic cardiovascular diseases opens a novel and challenging clinical option besides or in addition to...

8/3, K/14 (Item 6 from file: 5)
DIALOG(R) File 5:Biosis Previews(R)
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09004482 BIOSIS NO.: 199497012852

Human mesangial cells and *peripheral* *blood* mononuclear cells produce vascular permeability factor.

AUTHOR: Iijima Kazumoto(a); Yoshikawa Norishige; Connolly Daniel T;
Nakamura Hajime

AUTHOR ADDRESS: (a)Dep. Pediatrics, Kobe Univ. Sch. Med., 5-2 Kusunoki-cho
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JOURNAL: Kidney International 44 (5):p959-966 1993

ISSN: 0085-2538

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

Human mesangial cells and *peripheral* *blood* mononuclear cells produce vascular permeability factor.

ABSTRACT: Vascular permeability factor, or vascular endothelial growth factor (VPF/*VEGF*) is a disulfide-linked dimeric glycoprotein of about 40 kD that promotes fluid and protein leakage from blood vessels. Various human tumor cell lines and cells including fetal vascular smooth muscle cells produce VPF/*VEGF*. Since glomerular mesangial cells (MC) are closely related to vascular smooth muscle cells, we examined whether cultured human MC produce VPF/*VEGF*. Northern blotting analysis revealed that cultured human MC expressed a 3.7 kilobases (kb) VPFNEG mRNA. Human *peripheral* *blood* mononuclear cells (PBMC) also expressed VPF/*VEGF* transcripts of 8.6 and 3.8 kb. Although the sizes of the transcripts suggested the existence of unique molecular species of VPF/*VEGF* mRNA in PBMC, RT-PCR analysis revealed that PBMC as well as human MC expressed 121, 165, and 189 amino acid-containing isoforms of VPF/*VEGF*, implying that there are no unique alternative splicing products of VPF/*VEGF* mRNA in PBMC. Fetal calf serum and 12-o-tetradecanoyl-phorbol-13-acetate (TPA) transiently enhanced VPF/*VEGF* mRNA expression in cultured human MC. Transforming growth factor-beta-1 enhanced VPF/*VEGF* mRNA expression in cultured human MC at least within 24 hours. Dexamethasone (DEX) inhibited the TPA-induced increase in VPF/*VEGF* mRNA expression, whereas DEX did not change the basal level. That DEX depressed the TPA-induced increase in VPF/*VEGF* mRNA expression is therefore probably a result of transcriptional control. VPF/*VEGF* protein was detected in cultured human MC with immunoperoxidase staining using anti-VPF/*VEGF* antibody. TPA increased VPF/*VEGF* protein levels as well as those of VPF/*VEGF* mRNA in cultured human MC. These findings indicate that cultured human MC and PBMC produce VPF/*VEGF* and that it is modulated by

various agents. Since /*VEGF* promotes growth in vascular *endothelial* *cells* and enhances vascular permeability, VPF/ *VEGF* produced by MC and PBMC may induce the *proliferation* of glomerular *endothelial* *cells* or enhance the permeability of glomerular capillaries.

8/3,K/15 (Item 1 from file: 73)
DIALOG(R) File 73:EMBASE
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11092287 EMBASE No: 2001099239

Circulating *endothelial* *cells*: Realities and promises for new therapies of angiogenesis

LES PROGENITEURS ENDOTHELIAUX CIRCULANTS: REALITES ET PROMESSES POUR DE NOUVELLES THERAPIES DE L'ANGIOGENESE

Chagraoui J.; Uzan G.

G. Uzan, Inserm U. 506, Batiment Lavoisier, Hopital Paul-Brousse, 12, avenue Paul-Vaillant-Couturier, 94807 Villejuif Cedex France

Hematologie (HEMATOLOGIE) (France) 2001, 7/1 (68-77)

CODEN: HEMAF ISSN: 1264-7527

DOCUMENT TYPE: Journal ; Short Survey

LANGUAGE: FRENCH SUMMARY LANGUAGE: ENGLISH; FRENCH

NUMBER OF REFERENCES: 55

Circulating *endothelial* *cells*: Realities and promises for new therapies of angiogenesis

...hemangioblast. The existence of such hemangioblasts in adults was recently suggested, as a consequence of the discovery of circulating endothelial progenitor cells (angioblasts) in the *peripheral* *blood*. It is likely that these cells have a medullar origin. Different growth factors like *VEGF* or G-CSF can mobilize these cells into the blood circulation. These cells are functional, because they can be incorporated into the vessels formed in...

...grow slowly during about two weeks. The cells then acquire a very high proliferative potential, that could be maintained several weeks along. This kinetic of *proliferation* is different to that observed for *endothelial* *cells* from vessels, that display a lower proliferative potential. The physiological role of these angioblasts is still unclear. However, they display functional properties that are very...

...tissues. Second, recent studies have shown that in the bone marrow of patients with leukaemia, angiogenesis increases. Leukemic cells produce pro-angiogenic factors such as *VEGF*. The bcr/abl fusion gene is found in the majority of the haematopoietic cells of patients with chronic myeloid leukaemia (CML). This fusion gene was recently detected in the circulating *endothelial* *cells* of patients with CML, thus showing that the chromosomal translocation had occurred in medullar hemangioblasts. These observations indicate that medullar angiogenesis may play a role in the *proliferation* of leukemic cells. Inhibition of this angiogenesis may be a new strategy for the treatment of leukaemia. Finally, experiments performed in monkeys have shown that...

MEDICAL DESCRIPTORS:

precursor cell; cell motility; cell population; cell culture; cell *proliferation*; cell function; vascularization; ischemia; bone marrow; chromosome translocation; chronic myeloid leukemia; target cell; monkey; gene therapy; short survey

8/3,K/16 (Item 2 from file: 73)
DIALOG(R) File 73:EMBASE
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11075288 EMBASE No: 2001062740

Monocytes coexpress endothelial and macrophagocytic lineage markers and form cord-like structures in MatrigelSUP(R) under angiogenic conditions
Schmeisser A.; Garlichs C.D.; Zhang H.; Eskafi S.; Graffy C.; Ludwig J.;
Strasser R.H.; Daniel W.G.
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Cardiovascular Research (CARDIOVASC. RES.) (Netherlands) 16 FEB 2001
, 49/3 (671-680)
CODEN: CVREA ISSN: 0008-6363
PUBLISHER ITEM IDENTIFIER: S0008636300002704
DOCUMENT TYPE: Journal ; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 25

Objectives: It has been shown that circulating human non-adherent CD34SUP+ cells coexpressing vascular endothelial growth factor (*VEGF*)-R2 and AC133 have the capacity to differentiate into adherent mature *endothelial* *cells*. However, prior studies have demonstrated that a much bigger subset of primary adherent mononuclear cells can also form endothelial progenitor cells (EPC). To determine the...

...as a firmly adherent and plastic cell type have the potential to differentiate into an endothelial phenotype. Methods: CD34SUP-/CD14SUP+ monocytes were isolated from human *peripheral* *blood* by adherence separation and magnetic bead selection (purity >90%) and cultured on fibronectin-coated plastic dishes (medium containing *VEGF* 10 ng/ml, basic fibroblast growth factor (bFGF) 2 ng/ml, insulin like growth factor (IGF-1) 1 ng/ml, 20% fetal calf serum). Results...

MEDICAL DESCRIPTORS:

macrophage; endothelium cell; cell differentiation; cell culture; cell isolation; flow cytometry; cell *proliferation*; human; human cell; article ; priority journal
?ds

Set	Items	Description
S1	3580	(ENDOTHELIAL (W) CELLS) AND ((PERIPHERAL (W) BLOOD) OR (BU- FFY (W) COAT))
S2	96	S1 AND (VEGF)
S3	3	S2 AND (COLLAGEN)
S4	2	RD (unique items)
S5	0	S2 AND (BRAIN (W) EXTRACT)
S6	0	S2 AND (COBBLESTONE)
S7	36	S2 AND (PROLIFERATION OR EXPANSION)
S8	16	RD (unique items)

?logoff

04apr03 15:47:13 User259876 Session D485.2
\$2.51 0.786 DialUnits File155
\$1.68 8 Type(s) in Format 3
\$1.68 8 Types
\$4.19 Estimated cost File155
\$1.07 0.362 DialUnits File159
\$0.26 1 Type(s) in Format 3
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\$1.33 Estimated cost File159
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\$12.25 7 Types
\$16.53 Estimated cost File5
\$12.04 1.338 DialUnits File73
\$5.00 2 Type(s) in Format 3
\$5.00 2 Types
\$17.04 Estimated cost File73
OneSearch, 4 files, 3.250 DialUnits FileOS
\$2.32 TELNET
\$41.41 Estimated cost this search

\$41.88 Estimated to session cost 3.354 DialUnit

Status: Signed Off. (11 minutes)